the entire cohort immediately after the baseline measurement and is being followed up biennially for vital status. Follow-up for cancer was established by annual record linkages with the Netherlands Cancer Registry and the nationwide pathology registry. After 9.3 years of follow-up, 275 incident cases of renal cell cancer and 4779 subcohort members were available for analysis. Paraffin-embedded tumor tissue samples could be collected for 193 renal cell cancer patients from more than 50 pathology laboratories in the country. One pathologist (CAHK) classified all tumor samples morphologically. DNA from malignant tissue was isolated for *VHL* analysis. Samples were analyzed by SSCP, followed by direct sequencing when aberrant SSCP signals were detected. RRs and corresponding 95% confidence intervals (95% CI) for RCC, mutated clear-cell RCC and wildtype clear-cell RCC were estimated using Cox proportional hazard models. At baseline, cohort members who reported a history of hypertension or use of anti-hypertensive medication had a slightly increased risk of renal cell carcinoma: relative rate (RR) 1.32 (95% confidence interval (95% CI), 0.99-1.75) and 1.18 (95% CI, 0.86-1.62) respectively. RRs were adjusted for sex, age, weight, height and cigarette smoking. Of the 193 patients for whom tissue specimens could be collected, 153 had a clear cell renal cell carcinoma (CC-RCC, 79%). In 93 patients with CC-RCC a mutation in the *VHL*-gene was detected (61%). Hypertension or use of anti-hypertensive medication was associated with an increased risk of CC-RCC with *VHL* mutations: RR, 1.64 (95% CI, 1.02-2.63) and RR, 1.41 (95% CI, 0.82-2.41), respectively. For specific anti-hypertensive medication, beta-blocker use was associated with an increased risk of CC-RCC with *VHL* mutations: RR, 1.76 (95% CI, 0.98-3.14), while use of diuretics was not. Hypertension and use of anti-hypertensive medication were not associated with the risk of CC-RCC without *VHL* mutations; RR, 0.73 (95% CI, 0.39-1.37) and RR, 1.21 (95% CI, 0.65-2.23), respectively. This study confirmed the association of hypertension and –to a lesser extent- use of anti-hypertensive medication with renal cell cancer. These risk factors are associated mainly with RCC patients with *VHL* mutations, which suggests that the pathway from hypertension to RCC acts through mutations in the *VHL*-gene.

EPIDEMIOLOGY/LIFESTYLE FACTORS: Other Risk Factors

#A107 Significant Difference in Incidence Trend of Nasopharyngeal Carcinoma and Other Head and Neck Cancers for Men in Taiwan: an Age-Period-Cohort Analysis. Chiun Hsu,[1] Chee-Jen Chang,[1] Chia-Ling Kuo,[1] Ying-Chun Shen,[2] Ruey-Long Hong,[1] Ann-Lii (. Cheng.[1] National Taiwan University Hospital,[1] Taipei, Taiwan, Far Eastern Memorial Hospital,[2] Taipei, Taiwan.

The incidence of nasopharyngeal carcinoma (NPC, ICD9 code 147) for men in Taiwan was relatively stationary in the past 20 years (crude incidence 9.11 per 100,000 men in 1981 and 9.02 in 2000, respectively). On the other hand, the crude incidence of other head and neck cancers (HNC, ICD9 code 140 to 149, excluding 142 and 147) rose from 4.07 in 1981 to 27.04 in 2000. We compared the incidence trend of NPC and HNC for men in Taiwan from 1981 to 2000 by using the age-period-cohort model. Epidemiologic data were obtained from the Taiwan Cancer Registry. Age-specific incidences of NPC and HNC were plotted by calendar year at diagnosis and by birth cohort. The calendar time period 1986-1990 and the 1932-1940 birth cohort were used as reference groups for estimates of relative risk. For NPC, a negative cohort effect was found on the incidence trend, whereas the period effect was not significant. The relative risk of NPC for the 1972-1980 birth cohort was 0.69 (95% C.I. 0.51 to 0.94). By contrast, a strong positive cohort effect was noted for HNC. The relative risk of HNC for the 1972-1980 birth cohort was 45.67 (95% C.I. 28.68 to 72.74). A trend of a positive period effect was also found (relative risk 1.49, 95% C.I. 0.62 to 3.60). The strong cohort effect for HNC was correlated with a 6.85 fold increase of domestic production of betel quid, one of the most important risk factors for HNC, in the same period of time. Our results indicate NPC and HNC in Taiwan have different incidence trends because of different susceptibility of risk factors. Control of betel quid use will be a crucial issue for prevention of HNC. (Supported by grants NHRI-CN-CA9201S(93A059) and NTUH-93A14).

#A108 *Helicobacter pylori* and Risk of Gastric Cancer. Farin Kamangar,[1] Sanford Dawsey,[1] Guillermo Perez-Perez,[2] Pirjo Pietinen,[3] Craig J. Newschaffer,[4] Kathy Helzlsouer,[4] Yin Y. Shugart,[4] Christian C. Abnet,[1] Demetruis Albanes,[1] Jarmo Virtamo,[3] Philip R. Taylor.[1] National Cancer Institute,[1] Bethesda, MD, New York University,[2] New York, Finland National Public Health Institute,[3] Helsinki, Finland, Johns Hopkins Bloomberg School of Public Health,[4] Baltimore.

Although *Helicobacter pylori* (Hp) is considered a definite risk factor for gastric noncardia adenocarcinoma (GNCA), the magnitude of this association and the role of Hp in the etiology of histological subtypes of GNCA (intestinal vs. diffuse) are less clear. The association of Hp with gastric cardia adenocarcinoma (GCA) also remains unclear. We conducted a prospective nested case- control study to examine the association of Hp with risk of gastric cancer by its anatomical subsites and histological subtypes. Cases and controls were selected from the 29,133 male smokers aged 50-69 years who were recruited into the Alpha-Tocopherol, Beta-Carotene Cancer Prevention (ATBC) Study from 1985 to 1988. At baseline, a general background questionnaire and a detailed food frequency questionnaire were administered to each participant and baseline serum was collected. From 1985 to 1999, 243 cases of gastric adenocarcinoma (179 GNCA and 64 GCA) were diagnosed in this cohort. Serum samples from 234 gastric adenocarcinoma patients (173 GNCA and 61 GCA) and 234 age-matched controls were assayed for seropositivity to antibodies against whole cell Hp and CagA antigens. Hp was a strong risk factor for GNCA (adjusted odds ratio (OR) = 7.92, 95% confidence interval (95% CI) = (3.02 - 20.90)) but reduced the risk of GCA (OR = 0.31, 95% CI = (0.11 - 0.89)). Hp was an equally strong risk factor for intestinal and diffuse subtypes of GNCA. Time from serum collection to cancer occurrence did not modify the results. We conclude that Hp is a strong risk factor for GNCA but may be protective against GCA in this population.

#A109 A Prospective Investigation of Height and Prostate Cancer Risk in Male Smokers. Margaret E. Wright,[1] Jacqueline Sequoia,[1] Pirjo Pietinen,[2] Philip R. Taylor,[3] Jarmo Virtamo,[2] Demetrius Albanes.[1] Division of Cancer Epidemiology and Genetics, National Cancer Institute, NIH, DHHS,[1] Bethesda, MD, Department of Epidemiology and Health Promotion, National Public Health Institute,[2] Helsinki, Finland, Center for Cancer Research, National Cancer Institute, NIH, DHHS,[3] Bethesda, MD.

Increased adult height, a crude indicator of childhood nutritional status and growth hormone exposure, has been associated with prostate cancer risk in several observational studies, but the findings have been inconsistent. We examined the association between adult height and incident prostate cancer in the Alpha-Tocopherol, Beta-Carotene Cancer Prevention Study (ATBC) cohort. At baseline, 29,119 Finnish male smokers 50 to 69 years old had height and weight measured by trained personnel, provided information on demographic, smoking, and medical factors, and completed an extensive food frequency questionnaire. A total of 1,346 incident prostate cancer cases were identified during a follow-up period of up to 17.4 years (median = 14.1 years). In age-adjusted Cox proportional hazards models, the relative risks (RR) and 95% confidence intervals for prostate cancer according to increasing quintiles of height [cm; <168, 169-171, 172-175, 176-178, and 179-200] were 1.00 (referent), 1.11 (0.93-1.32), 1.11 (0.95-1.31), 1.30 (1.01-1.55), and 1.14 (0.96-1.35), P trend = 0.05). In analyses stratified by disease stage (available for 916 (68 %) of the cases), a strong linear association between height and prostate cancer risk was observed among those with advanced lesions (TNM stage >3, RR for highest versus lowest quintile of height = 2.02 (1.37 – 2.97), P trend = 0.0008), but not among those with less aggressive disease (P interaction= 0.002). Our study provides additional evidence that increased height is a significant risk factor for prostate cancer, and suggests that taller men are particularly susceptible to advanced disease.

#A110 The Prevalence of Hepatitis C Infection in Patients of Non-Hodgkin's Lymphoma in British Columbia: A Case-Control Study. Agnes S. Lai,[1] John J. Spinelli,[1] Randy D. Gascoyne,[1] Joseph M. Connors,[1] Anton P. Andonov,[2] Darrel Cook,[3] Pat Lee,[1] Rozmin Janoo-Galani,[1] Richard Gallagher.[1] BC Cancer Agency,[1] Vancouver, BC, Canada, Health Canada, National Microbiology Laboratories,[2] Winnipeg, Manitoba, Canada, British Columbia Centre for Disease Control,[3] Vancouver, British Columbia, Canada.

Background: Hepatitis C virus (HCV) is a major cause of chronic liver diseases and of mixed cryoglobulinemia, and it has been found to be